

value, a maternal BPb value of 10 µg/dl, as seen in Table 1, should not be exceeded. If no more than 5% of breast-feeding infants are to exceed the 10 µg/dl action level, then a maternal BPb <10 µg/dl is required.

A sensitivity analysis using various IEUBK modeling runs shows that the infant body lead burden at birth, from *in utero* accumulation via maternal lead exposures, is mainly expressed through estimated BPb values in the first 6 months of infant life, as compared to the second 6 months of infant life or as compared to exposure integrated over the entire first year of infant life. This is to be expected, given the relatively high biokinetic mobility of lead in the very young. However, it is precisely in the first 6 months of infant life that breast-feeding is done. Therefore, both breast milk lead and prior infant body lead burden are significant sources of lead in breast-feeding infants of mothers with elevated lead exposures. In essence, the only maternal BPb level that is in fact "safe" in terms of CDC Class III elevated infant BPb figures also approximates the CDC infant BPb action level of 10 µg/dl. In terms of the child action level of 10 µg/dl, a maternal BPb <10 µg/dl appears prudent.

The 1991 CDC statement on childhood lead poisoning (6) identified a BPb level of 10 µg/dl as being the body lead threshold associated with the earliest toxic effects in infants and toddlers. The CDC document also accepted the risk assessment premise that there is no known threshold for lead's subtle toxicity.

Sinks and Jackson argue that the most recent NHANES III, 1991–1994, indicates that there are no women in the United States who are likely to be nursing their infants and who have BPb values anywhere close to the 40 µg/dl Sinks and Jackson claimed as permissible for nursing mothers. They cite some actual numbers noted in the NHANES III data tapes (7). Such prevalence data are aggregated cluster sample depictions at a single time point of the U.S. population lead exposure picture, stratified by national socioeconomic

and demographic strata. One cannot legitimately disaggregate such national depictions or "snapshots" to generate comparisons for individual community prevalences or to use actual BPb values contained in any particular statistical sampling cell in the aggregation process. Such limits are discussed in, among other things, the Executive Summary of the 1988 report to the U.S. Congress on childhood lead poisoning (8) by the Agency for Toxic Substances and Disease Registry.

Sinks and Jackson offer the simplistic and incorrect argument that if women in the United States were nursing their infants and had elevated BPbs from workplace exposures, they would be readily and reliably detected by Occupational Safety and Health Administration (OSHA)-required exposure and medical surveillance. Many small operations are either exempt from OSHA requirements because of size or are rarely if ever inspected because of severe resource constraints on federal or various state OSHA agencies. Such assessments similarly would not detect the women who potentially have elevated BPb values owing to environmental, not occupational, lead exposures. A much better approach, regardless of sources of high maternal exposure, would be the approach endorsed by Lawrence (3): maternal/infant screening.

The letter by Sinks and Jackson may be seen by some as another example of an overall CDC retreat from lead as a persisting child health issue. Needleman (9) noted an overall backpedaling in efforts and decline in momentum to finally address the lead issue in a meaningful way by the federal government. All this raises a legitimate question among scientists and clinicians interested in lead: Is lead still considered to be a child health risk issue at the CDC or elsewhere in the federal government?

The actual content of new research should be understood before wholesale attacks on such research are launched. This is especially the case where complex research designs and equally complex results are at work. In those cases where breast milk does not contain worrisome lead concentrations

from high maternal lead exposures, per Table 1, I agree with the Sinks and Jackson comment that "breast is best."

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## Response to Sinks and Jackson

We appreciate Sinks' and Jackson's interest in our article on lead in breast milk and would like to reinforce and clarify a couple of points. Paul Mushak has responded comprehensively to their letter, and we are in complete agreement with his response.

We agree wholeheartedly that "breast is best," and our low concentrations of lead in breast milk confirm this. Our abstract (1) was quite emphatic that

Breast-fed infants are only at risk if the mother is exposed to high concentrations of contaminants either from endogenous sources such as the skeleton or exogenous sources.

Sinks and Jackson are dismissive of the use of lead isotopic ratios as not being "meaningful in establishing risk for lead poisoning." Perhaps this is true in the strict sense of risk assessment, but lead isotopic ratios are the only realistic method of determining the source of a mother's lead burden.

**Table 1.** Predicted blood lead (BPb) levels in breast-feeding infants at various maternal BPb levels<sup>a</sup>

| Maternal nursing BPb (µg/dl) <sup>b</sup> | Infant Pb intake (µg/day) <sup>c</sup> | GM infant BPb (µg/dl) <sup>d</sup> | % >10 µg/dl <sup>e</sup> | % >20 µg/dl <sup>f</sup> |
|---|--|------------------------------------|--------------------------|--------------------------|
| 40  | 48                                     | 19.4                               | 90.7                     | 45.1                     |
| 30  | 36                                     | 15.4                               | 78.4                     | 26.9                     |
| 20  | 24                                     | 11.3                               | 58.3                     | 10.6                     |
| 15  | 18                                     | 9.1                                | 40.4                     | 4.4                      |
| 10  | 12                                     | 6.9                                | 19.8                     | 1.0                      |

GM, geometric mean.

<sup>a</sup>EPA's Integrated Exposure-Uptake Biokinetic Model, Version 0.99d; input parameters were milk Pb uptake = 50%; 0 ml tap water; milk Pb = 100% diet + fluid Pb; model default dust/soil inputs for 0–12-month-old infants.

<sup>b</sup>Breast milk feeding period, 0–6 months of age; maternal BPb levels are present at birth through 6 months.

<sup>c</sup>Infant Pb intake/day = 0.15 × maternal BPb (µg/dl) × 8 dl milk/day.

<sup>d</sup>Geometric standard deviation = 1.6; GM infant BPb and percentages exceeding cutoffs obtained from graphic outputs expressed as probability density function histograms versus infant BPb and using age band "A" (0–6 months of age).

<sup>e</sup>CDC action level of ≥10 µg/dl.

<sup>f</sup>CDC Class III, medical intervention level of ≥20 µg/dl.

Sinks and Jackson also appear dismissive of the recommendation of screening women for lead body burden via blood lead levels. We stress that this would be only for those women that were exposed in the past or are currently exposed to lead. We would certainly extend any screening to women employed in lead-exposed jobs as they cite. However, the overwhelmingly vast majority of women (and their partners and infants) at risk are those exposed during house renovations involving leaded paint, from dust from ceiling and wall cavities, from "take-home" dust from their jobs, from hobbies, etc.; this can affect all socioeconomic levels. In fact, the most disturbing aspect of lead poisoning from do-it-yourself activities is that most of these people are unaware of the dangers.

No action level was given in our paper because there are no accepted guidelines for blood lead levels and breast-feeding apart from the Lawrence document (2) noted by Sinks and Jackson, the relevance of which has been questioned by Mushak in his letter. In the absence of guidelines, we recommend

that if the mother-to-be is concerned that she may have been heavily exposed to lead from any source at any time, she request a blood lead measurement either before conception or at least during the first trimester. If the blood lead level is greater than two times the CDC "level of concern" (i.e., >20 µg/dl), we recommend that she have her breast milk tested by a reputable laboratory.

In addition, we suggest that mothers-to-be maintain healthy diets and consume the NIH recommended daily intake of calcium of 1,100–1,200 mg Ca/day during pregnancy and up to 1,400 mg Ca/day during breast-feeding (3). This will not only potentially lessen the mobilization of endogenous lead from the maternal skeleton as shown in our recent studies (4,5) but also lessen the uptake of exogenous lead from the gastrointestinal tract.

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## REFERENCES AND NOTES

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## Corrections and Clarifications

In the article by Loewenherz et al. (Biological Monitoring of Organophosphorus Pesticide Exposure among Children of Agricultural Workers in Central Washington State) published in *EHP* [105:1344–1353 (1997)], two errors were discovered in the Appendix. The units, as they appear in the Appendix Tables 1, 2, and 3 are incorrect; the correct units are milligrams per gram creatinine. Also, several numbers in Table A.2 were not adjusted for creatinine. The corrected table appears below.

**Table A. 2.** Creatinine-adjusted dimethylthiophosphate (DMTP) levels (mg/g creatinine) in applicator and reference children for each separate visit<sup>a</sup>

|                                    | Applicator children |           | Reference children |          |
|------------------------------------|---------------------|-----------|--------------------|----------|
|                                    | Visit 1             | Visit 2   | Visit 1            | Visit 2  |
| All children                       |                     |           |                    |          |
| Mean <sup>b</sup>                  | 0.076               | 0.118     | 0.046              | 0.040    |
| Median                             | 0.016               | 0.045     | 0.000              | 0.000    |
| CV <sup>c</sup>                    | 211%                | 232%      | 267%               | 150%     |
| Range                              | ND–0.768            | ND–2.006  | ND–0.493           | ND–0.190 |
| Frequency <sup>d</sup>             | 23 (38)             | 35 (58)   | 3 (19)             | 6 (38)   |
| Number                             | 61                  | 60        | 16                 | 16       |
| Focus children (one per household) |                     |           |                    |          |
| Mean                               | 0.096               | 0.094     | 0.054              | 0.026    |
| Median                             | 0.035               | 0.045*    | 0.000              | 0.000*   |
| CV                                 | 188%                | 134%      | 254%               | 146%     |
| Range                              | ND–0.768            | ND–0.518  | ND–0.493           | ND–0.113 |
| Frequency                          | 21 (46)             | 24 (56)** | 3 (23)             | 4 (33)** |
| Number                             | 46                  | 43        | 13                 | 12       |

Abbreviations: CV, coefficient of variation; ND, not detectable.

<sup>a</sup>Tests for statistical significance applied to focus children data only (see Methods).

<sup>b</sup>Mean and other univariate statistics were calculated by estimating trace samples as 1/2 limit of detection.

<sup>c</sup>CV = standard deviation/mean × 100.

<sup>d</sup>Percent is shown in parentheses.

\*Significant difference across applicator and reference children:  $p = 0.038$  (Mann-Whitney  $U$  test).

\*\*Marginally significant difference across groups:  $p = 0.064$  (chi-square test).